What Is Claimed Is:

1. An ApoA-I agonist comprising:

(i) a 14 to 22-residue peptide or peptide analogue which forms an amphipathic α-helix in the presence of lipids and which comprises the structural formula (I):

Z_1-X_1-X_2-X_3-X_4-X_5-X_6-X_7-X_8-X_9-X_{10}-X_{11}-X_{12}-X_{13}-X_{14}-X_{15}-X_{16}-X_{17}-X_{18}

X_1 is Pro (P), Ala (A), Gly (G), Asn (N), Gln (Q) or D-Pro (p);

X_2 is an aliphatic amino acid;

X_3 is Leu (L);

X_4 is an acidic amino acid;

X_5 is Leu (L) or Phe (F);

X_6 is Leu (L) or Phe (F);

X_7 is a basic amino acid;

X_8 is an acidic amino acid;

X_9 is Leu (L) or Trp (W);

X_{10} is Leu (L) or Trp (W);

X_{11} is an acidic amino acid or Asn (N);

X_{12} is an acidic amino acid;

X_{13} is Leu (L), Trp (W) or Phe (F);

X_{14} is a basic amino acid or Leu (L);

X_{15} is Gln (Q) or Asn (N);

X_{16} is a basic amino acid;

X_{17} is Leu (L);

X_{18} is a basic amino acid;

Z_1 is H_2N- or RC(O)NH-;

Z_2 is -C(O)NRR, -C(O)OR or -C(O)OH or a salt thereof;

each R is independently -H, (C_1-C_6) alkyl, (C_1-C_6) alkenyl, (C_1-C_6) alkynyl, (C_5-C_{20}) aryl, (C_6-C_{26}) alkaryl, 5-20 membered heteroaryl or 6-26 membered alkheteroaryl or a 1 to 4-residue peptide or peptide analogue;

each " - " between residues X_n independently designates an amide linkage, a substituted amide linkage, an isostere of an amide or an amide mimetic; or

(ii) a deleted from of structural formula (I) in which at least one and up to eight of residues X_1, X_2, X_3, X_4, X_5, X_6, X_7, X_8, X_9, X_{10}, X_{11}, X_{12}, X_{13}, X_{14}, X_{15}, X_{16}, X_{17} and X_{18} are deleted; or

(iii) an altered form of structural formula (I) in which at least one of residues X_1, X_2, X_3, X_4, X_5, X_6, X_7, X_8, X_9, X_{10}, X_{11}, X_{12}, X_{13}, X_{14}, X_{15}, X_{16}, X_{17} or X_{18} is conservatively substituted with another residue.

2. The ApoA-I agonist of Claim 1 which exhibits at least about 38% LCAT-activation activity as compared with human ApoA-I.

3. The ApoA-I agonist of Claim 1 which is the altered form of structural formula (I).

4. The ApoA-I agonist of Claim 3 in which the hydrophobic residues are fixed according to structural formula (I) and at least one non-fixed residue is conservatively substituted with another residue.

5. The ApoA-I agonist of Claim 4 in which:

X_1 is Pro (P), D-Pro (p), Gly (G), Asn (N) or Ala (A);

X_2 is Ala (A), Leu (L) or Val (V);

X_3 is Leu (L);

X_5 is Leu (L) or Phe (F);

X_6 is Leu (L) or Phe (F);

X_9 is Leu (L) or Trp (W);

X_{10} is Leu (L) or Trp (W);

X_{13} is Leu (L), Trp (W) or Phe (F);

X_{17} is Leu (L); and

at least one of X_4, X_7, X_8, X_{11}, X_{12}, X_{14}, X_{15}, X_{16} and X_{18} is conservatively substituted with another residue.

6. The ApoA-I agonist of Claim 3 in which the hydrophilic residues are fixed according to structural formula (I) and at least one non-fixed residue is conservatively substituted with another residue.

7. The ApoA-I agonist of Claim 6 in which:

X_4 is Asp (D) or Glu (E);

X_7 is Arg (R), Lys (K) or Orn;

X_8 is Asp (D) or Glu (E);

X_{11} is Asn (N) or Glu (E);

X_{12} is Glu (E);

X_{14} is Lys (K), Arg (R) or Orn;

X_{15} is Gln (Q) or Asn (N);

X_{16} is Lys (K), Arg (R) or Orn;

X_{18} is Asn (N) or Gln (Q); and

at least one of X_1, X_2, X_3, X_5, X_6, X_9, X_{10}, X_{13} and X_{17} is conservatively substituted with another residue.

8. The ApoA-I agonist of Claim 6 in which X_3 is Leu (L), X_6 is Phe (F), X_9 is Leu (L) or Trp (W), X_{10} is Leu (L) or Trp (W) and at least one of X_1, X_2, X_5, X_{13} and X_{17} is conservatively substituted with another residue.

9. The ApoA-I agonist of Claim 5 or 7 in which the substituting residue is classified within the same sub-category as the substituted residue.

10. The ApoA-I agonist of Claim 1 which is the deleted form of structural formula (I).

11. The ApoA-I agonist of Claim 10 in which one helical turn of the peptide or peptide analogue is deleted.

12. The ApoA-I agonist of Claim 1 which is an 18-residue peptide or peptide analogue of structural formula (I).

13. The ApoA-I agonist of Claim 12 in which:
the "-" between residues designates -C(O)NH-;
Z_1 is H_2N-; and
Z_2 is -C(O)OH or a salt thereof.

14. The ApoA-I agonist of Claim 13, in which:
X_1 is Pro (P), Ala (A), Gly (G), Asn (N) or D-Pro (p);
X_2 is Ala (A), Val (V) or Leu (L);
X_3 is Leu (L);
X_4 is Asp (D) or Glu (E);
X_5 is Leu (L) or Phe (F);
X_6 is Leu (L) or Phe (F);
X_7 is Arg (R), Lys (K) or Orn;
X_8 is Asp (D) or Glu (E);
X_9 is Leu (L) or Trp (W);
X_{10} is Leu (L) or Trp (W);
X_{11} is Glu (E) or Asn (N);
X_{12} is Glu (E);
X_{13} is Leu (L), Trp (W) or Phe (F);
X_{14} is Arg (R), Lys (K) or Orn;
X_{15} is Gln (Q) or Asn (N);
X_{16} is Arg (R), Lys (K) or Orn;
X_{17} is Leu (L); and
X_{18} is Arg (R), Lys (K) or Orn.

15. The ApoA-I agonist of Claim 1 which is selected from the group consisting of:

peptide **191** PVLDLLRELLEELKQKLK* (SEQ ID NO:191);
peptide **192** PVLDLFKELLEELKQKLK* (SEQ ID NO:192);
peptide **193** PVLDLFRELLEELKQKLK* (SEQ ID NO:193);
peptide **194** PVLELFRELLEELKQKLK* (SEQ ID NO:194);

peptide **195** PVLELFKELLEELKQKLK* (SEQ ID NO:195);
peptide **196** PVLDLFRELLEELKNKLK* (SEQ ID NO:196);
peptide **197** PLLDLFRELLEELKQKLK* (SEQ ID NO:197);
peptide **198** GVLDLFRELLEELKQKLK* (SEQ ID NO:198);
peptide **199** PVLDLFRELWEELKQKLK* (SEQ ID NO:199);
peptide **200** NVLDLFRELLEELKQKLK* (SEQ ID NO:200);
peptide **201** PLLDLFKELLEELKQKLK* (SEQ ID NO:201);
peptide **202** PALELFKDLLEELRQKLR* (SEQ ID NO:202);
peptide **203** AVLDLFRELLEELKQKLK* (SEQ ID NO:203);
peptide **204** PVLDFFRELLEELKQKLK* (SEQ ID NO:204);
peptide **205** PVLDLFREWLEELKQKLK* (SEQ ID NO:205);
peptide **206** PLLELLKELLEELKQKLK* (SEQ ID NO:206);
peptide **207** PVLELLKELLEELKQKLK* (SEQ ID NO:207);
peptide **208** PALELFKDLLEELRQRLK* (SEQ ID NO:208);
peptide **209** PVLDLFRELLNELLQKLK (SEQ ID NO:209);
peptide **210** PVLDLFRELLEELKQKLK (SEQ ID NO:210);
peptide **211** PVLDLFRELLEELOQOLO* (SEQ ID NO:211);
peptide **212** PVLDLFOELLEELOQOLK* (SEQ ID NO:212);
peptide **213** PALELFKDLLEEFRQRLK* (SEQ ID NO:213);
peptide **214** pVLDLFRELLEELKQKLK* (SEQ ID NO:214);
peptide **215** PVLDLFRELLEEWKQKLK* (SEQ ID NO:215);
peptide **229** PVLELFERLLEDLQKKLK (SEQ ID NO:229);
peptide **230** PVLDLFRELLEKLEQKLK (SEQ ID NO:230);
peptide **231** PLLELFKELLEELKQKLK* (SEQ ID NO:231);

in either the N- and/or C-terminal blocked or unblocked forms.

16. A multimeric ApoA-I agonist which exhibits at least about 38% LCAT activation activity as compared with human ApoA-I and which has the structural formula (II):

(II) $HH\{LL_m\text{-}HH\}_nLL_m\text{-}HH$

or a pharmaceutically acceptable salt thereof, wherein:

each m is independently an integer from 0 to 1;

n is an integer from 0 to 10;

each "HH" is independently a peptide or peptide analogue according to Claim 1;

each "LL" is independently a bifunctional linker; and

each " - " independently designates a covalent linkage.

17. A multimeric ApoA-I agonist which exhibits at least about 38% LCAT activation activity as compared with human ApoA-I and which has the structural formula (III):

(III) $X\text{-}N_{ya}\text{-}X_{(ya-1)}\text{-}(N_{yb}\text{-}X_{(yb-1)})_p$

or a pharmaceutically acceptable salt thereof, wherein:

each X is independently $HH\text{-}(LL_m\text{-}HH)_nLL_m\text{-}HH$;

each HH is independently a core peptide of structure (I) or an analogue or mutated, truncated, internally deleted or extended form thereof as described herein;

each LL is independently a bifunctional linker;

each m is independently an integer from 0 to 1;

each n is independently an integer from 0 to 8;

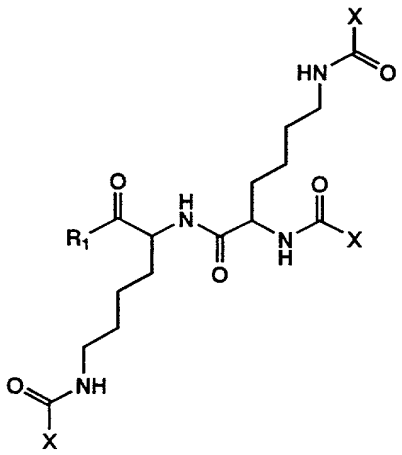
N_{ya} and N_{yb} are each independently a multifunctional linking moiety where y_a and y_b represent the number of functional groups on N_{ya} and N_{yb}, respectively;

each y_a or y_b is independently an integer from 3 to 8;

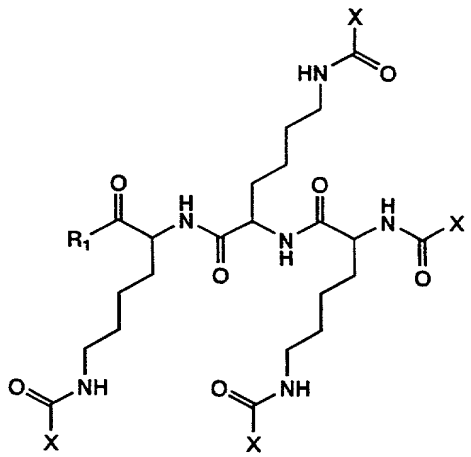
p is an integer from 0 to 7; and

each "—" independently designates a covalent bond.

18. A multimeric ApoA-I agonist which exhibits at least about 38% LCAT activation activity as compared with human ApoA-I and which has the structural formula (IV) or (V):



-OR-



(IV) (V)

or a pharmaceutically acceptable salt thereof, wherein:

each X is independently $HH\{LL_m—HH\}_nLL_m—HH$;

each HH is independently a peptide or peptide analogue according to Claim 1;

each LL is independently a bifunctional linker;

each n is independently an integer from 0 to 1;

each m is independently an integer from 0 to 8;

R_1 is -OR or -NRR; and

each R is independently -H, (C_1-C_6) alkyl, (C_1-C_6) alkenyl, (C_1-C_6) alkynyl; (C_5-C_{20}) aryl (C_6-C_{26}) alkaryl, 5-20 membered heteroaryl or 6-26 membered alkheteroaryl.

19. The multimeric ApoA-I agonist of Claim 16, 17 or 18 in which the bifunctional linker is cleavable.

20. The ApoA-I multimeric agonist of Claim 16, 17 or 18 in which n is 0.

21. The multimeric ApoA-I agonist of Claim 20 in which m is 0.

22. The multimeric ApoA-I agonist of Claim 16, 17 or 18 in which each HH is independently a peptide according to Claim 13.

23. The multimeric ApoA-I agonist of Claim 16, 17 or 18 in which each HH is independently a peptide according to Claim 14.

24. The multimeric ApoA-I agonist of Claim 16, 17 or 18 in which each HH is independently a peptide according to Claim 15.

25. An ApoA-I agonist-lipid complex comprising an ApoA-I agonist and a lipid, wherein the ApoA-I agonist is a peptide or peptide analogue according to Claim 1, a multimeric ApoA-I agonist according to Claim 16, a multimeric ApoA-I agonist according to Claim 17, or a multimeric ApoA-I agonist according to Claim 18.

26. The ApoA-I agonist-lipid complex of Claim 25 in which the ApoA-I agonist is a peptide according to Claim 12.

27. The ApoA-I agonist-lipid complex of Claim 25 in which the ApoA-I agonist is a peptide according to Claim 13.

28. The ApoA-I agonist-lipid complex of Claim 25 in which the ApoA-I agonist is a peptide according to Claim 14.

29. The ApoA-I agonist-lipid complex of Claim 25 in which the ApoA-I agonist is a peptide according to Claim 15.

30. The ApoA-I agonist-lipid complex of Claim 25 in which the lipid is sphingomyelin.

31. The ApoA-I agonist-lipid complex of Claim 25 which is in the form of a lyophilized powder.

32. The ApoA-I agonist-lipid complex of Claim 25 which is in the form of a solution.

33. A pharmaceutical composition comprising an ApoA-I agonist and a pharmaceutically acceptable carrier, excipient or diluent, wherein the ApoA-I agonist is a peptide or peptide analogue according to Claim 1, a multimeric ApoA-I agonist according to Claim 16, a multimeric ApoA-I agonist according to Claim 17, or a multimeric ApoA-I agonist according to Claim 18.

34. The pharmaceutical composition of Claim 33 in which the ApoA-I agonist is a peptide according to Claim 12.

35. The pharmaceutical composition of Claim 33 in which the ApoA-I agonist is a peptide according to Claim 13.

36. The pharmaceutical composition of Claim 33 in which the ApoA-I agonist is a peptide according to Claim 14.

37. The pharmaceutical composition of Claim 33 in which the ApoA-I agonist is a peptide according to Claim 15.

38. The pharmaceutical composition of Claim 33, 34, 35, 36 or 37, in which the ApoA-I agonist is in the form of an ApoA-I agonist-lipid complex, said complex comprising the ApoA-I agonist and a lipid.

39. The pharmaceutical composition of Claim 38 in which the ApoA-I agonist-lipid complex is in the form of a lyophilized powder.

40. A method of treating a subject suffering from a disorder associated with dyslipidemia, said method comprising the step of administering to the subject an effective amount of the ApoA-I agonist of Claim 1.

41. The method of Claim 40 in which the ApoA-I agonist is in the form of a pharmaceutical composition, said composition comprising the ApoA-I agonist and a pharmaceutically acceptable carrier, excipient or diluent.

42. The method of Claim 40 in which the ApoA-I agonist is in the form of an ApoA-I agonist-lipid complex, said complex comprising the ApoA-I agonist and a lipid.

43. The method of Claim 40 in which the disorder associated with dyslipidemia is hypercholesterolemia.

44. The method of Claim 40 in which the disorder associated with dyslipidemia is cardiovascular disease.

45. The method of Claim 40 in which the disorder associated with dyslipidemia is atherosclerosis.

46. The method of Claim 40 in which the disorder associated with dyslipidemia is restenosis.

47. The method of Claim 40, in which the disorder associated with dyslipidemia is HDL or ApoA-I deficiency.

48. The method of Claim 40, in which the disorder associated with dyslipidemia is hypertriglyceridemia.

49. The method of Claim 40, in which the disorder associated with dyslipidemia is metabolic syndrome.

50. A method of treating a subject suffering from septic shock, said method comprising the step of administering to the subject an effective amount of the ApoA-I agonist of Claim 1.

51. The method of Claim 40 or 50 in which said subject is a human.

52. The method of Claim 40 or 50 in which about 0.5 mg/kg to about 100 mg/kg ApoA-I agonist is administered to said subject.